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Case-Report Open Access

Hypocalcemia Induced Severe Heart Insufficiency and Visual Acuity Impairment– Will Calcium Supplementation Improve Heart and Eye Function?

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Abstract

Introduction: Hypocalcemia is a potentially life-threatening biochemical disorder often misdiagnosed and mistreated.

Case report: A case of a female patient with unilateral visual acuity drop, significant neurological and cardiologic problems caused by severe hypocalcemia is presented. Supplementation therapy led to withdrawal of neurological symptoms, improvement of the heart function and stabilization of the left eye visual acuity. Right eye visual acuity was excellent with occasional drops correlating to low serum calcium level.

Discussion: Physicians should be aware of hypocalcemia because time between the onset of symptoms and diagnosis averages ten years and adequate treatment can prevent severe eye, heart and neurological complications. Hopefully, this case would be instructive for future medical practice, especially for recognition and treatment of the extremely rare case of unilateral hypocalcemia-induced optic neuritis.

Keywords: Hypocalcemia; Heart insufficiency; Visual acuity impairment

Introduction

Hypocalcemia is a potentially life-threatening biochemical disorder often misdiagnosed and mistreated. Most common cause of hypocalcemia is idiopathic hypoparathiroidism. The main characteristics of idiopathic hypoparathirodism are low serum calcium and elevated inorganic phosphorus of the serum [1]. Time between the onset of symptoms and diagnosis averages ten years [2]. There are several characteristics of hypocalcemia: bilateral cataracts, basal ganglia calcification, nephrocalcinosis, papilledema or optic neuritis and heart disease [3].

Hypocalcemia and papilledema leading to the loss of vision were first reported in 1903 and since then approximately fifty cases had been recorded [4]. Only three cases reported the occurrence of optic neuritis in patients with hypocalcemia [5-7]. All of these cases were bilateral and optic neuritis was preceded by papilledema. Subsequently, optic nerve head atrophied.

Case Report

A 52-year old female patient presented to ophthalmology practice with an unexplainable visual acuity on the left eye of 1/192. Pupillary reaction was sluggish with no relative afferent pupillary defect. Fundus examination revealed slightly paler optic disc with sharp margins. Macula and periphery were normal in appearance. A cataract surgery was performed on the same eye in February 2010. On the regular check-up's 1 and 4 weeks following the surgery her visual acuity remained 6/6.

Contrast sensitivity (Pelli-Robson chart) and color vision (Ishihara plates, Farnsworth D15 and Hua 100) were grossly decreased on both eyes, left more than right. Pattern VEP (Pattern Visually Evoked Potential) showed bilateral lower amplitudes of N1-P1 and P1-N2 (normal values 5-10 μV), more significant on the left eye and latency of P100 wave within normal limits (100 ± 10 ms). Flesh VEP (Flesh Visually Evoked Potential) showed bilateral lower amplitudes of N1-P1 and P1-N2, again more significant on the left eye and prolonged latency of P100 wave. Cerebrospinal fluid opening pressure and composition were normal. These findings, together with blind spot enlargement on the Humphrey perimetry, indicate bilateral lesion of the optic nerve more significant on the left eye.

Table 1, provides detailed medical history of the patient from the first symptoms which appeared in year 2006 until the day hypocalcemia-related optic neuritis was diagnosed and confirmed; Detailed patient's medical history from the onset of first symptoms until the moment of the diagnosis

Evidently, patient was misdiagnosed and mistreated several times, even receiving psychiatric therapy which is well known to further reduce serum calcium levels. At the moment of diagnosis, patient was astenic, with edematous legs, tetany, positive Chvostek's and Trousseau sign, almost blind from bilateral cataracts. Her life quality was extremely low and further decreasing; weight loss was substantial over 20 kg, ECG (Echocardiogram) indicated prolonged QT interval of 480 ms, with the left heart enlargement (Figure 1).

2006	2007	2008	2009	2010
First episode of chest suffocation.	More frequent episodes of chest suffocation.	Acralparastesia, then calf parestesia;	Still undergoing psychiatric therapy (Zoloft and Xalol) which are believed to further reduce Ca levels.	PTH low; T4, TSH, ANA, ANCA, anti-endomisial At, anti-TG, anti-TPOAT - normal. Anti-parietal At increased
Weight loss	further weight loss	Chest suffocation attack-diagnosed as COPD- received bronchodilatators	June 2009 tetany attacks during night.	Low levels of whole and ionized calcium, increased phosphate level.
			Extreme muscle pains in the arms as if though the arms were in the presses and carpal spasms – TROUSSEAU SIGN	
	Anxiety and mood swings. Therapy anaphranyl and lorozepam made her sleepy, dizzy and drugged	Whole body cramps and stiffness during one minute she couldn't move or breathe – TETANY	Extreme back pain, inability to breathe at night unless sat in upright position, leg edema extreme tiredness, she was unable to make more than ten steps without pause	Osteodensitometry within normal limits.
			Subsequent medical checkup showed pleural effusion up to 2/3 of the lungs and enlarged hart silhouette	ECHO of the abdomen and tyreoid gland within normal limits
				MRI showed calcification of the basal ganglia.
		During ECG another tetany-misdiagnosed as a hysteria attack – therapy Zoloft and Xalol, made hallucinate and experience persecution mania	Heart ECHO indicated dilatated cardiomyopathy, EF 37%, pericardium delamination up to 5 mm	Hypocalcemia due to idiopathic parathyreoidism was diagnosed
			ECG verified negative T-wave in D1, D1, AVL, V1 – V6, QTc was 378 ms	Substitutionalteraphy Calcium tbl. 1000+500+1000 mg and tbl. Rocatrol 2 x 0.5 mg
			She was given cardiology therapy (cornilat, cardiopirin and Lasix) +psychiatric treatment	
			Visual acuity drop-forced to use a magnifying glass for reading – bilateral cataract.	Left eye cataract operated February 2010, and released on the first postoperative day with visual acuity 20/20
			CK 25 times higher than normal High CK, LDH and transaminase levels-myositis was suspected - referred to a neurologist. Positive Chvostek and Trousseau signs-analysis of serum calcium	October-noticed first signs of the visual impairment
			Hypocalcemia diagnosed. She was referred to endocrinologist.	December 2010. VA dropped to 1/20. Optic neuritis was diagnosed

Table 1: Detailed patient's medical history from the onset of first symptoms until the moment of the diagnosis.

The patient's heart echo showed severe heart insufficiency with dilatation of left ventricle and atrium. Ejection fraction was 37% with delamination of pericardium 5 mm (Table 2). She had extreme pleural effusion of 2/3 of the lungs. With adequate substitutional therapy almost all neurological problems withdrew. Even the heart function improved significantly during a three-year therapy period, heart insufficiency improving from 3+ to 1+ with ejection fraction of 60% (Table 2)

At the moment of postoperative left eye visual acuity reduction the patient's serum calcium was normal (Table 2). Using substitutional calcium and vitamin D therapy serum calcium was maintained within normal range. Visual acuity drop on patient's right eye was significant so it was decided to operate cataract in April 2011 in order to find out whether it was due to cataract or to optic neuritis. Postoperative visual acuity was excellent 6/6.

Several months later on the regular checkup the patient reported temporary vision reductions which were correlated to low calcium serum levels. In order to examine the relationship between the visual acuity drop and serum calcium levels, the patient was ordered to report to practice each time such temporary visual acuity loss occurred. Visual acuity was tested on Snellen and ETDRS (Early Treatment Diabetic Retinopathy Study) chart and blood samples were taken for analysis. As indicated in the presented table, the patient's visual acuity on the right, healthy eye was reduced during the moments of low serum calcium levels.

Discussion

This study presents a patient with idiopathic hypoparathyriodism who was diagnosed three and a half years from the appearance of first symptoms which is far below average of ten years.

This case is unique due to its unilaterality and, although the patient reported to the practice fairly quickly (one month after the noticeable visual acuity loss), it was not possible to find swollen PNO (Papilla nervi optici-optic nerve head).

Ayuk et al. proposed vascular pathogenesis (Anterior Ischemic Optic Neuropathy, AION), as a cause of visual loss in a patient with hypoparathyrodism [8].

Mukhopadhyay [9] reported a case of hypocalcemia with simultaneous papilledema and optic atrophy (pseudo-Foster Kennedy syndrome). In this case initiation of calcium therapy lead to improvement of the optic neuropathy. He also suggested that treatment should be started early to prevent optic atrophy. In the case presented in this report visual acuity drop occurred in the moment of the well-corrected level of serum calcium.

As McLean et al. [4] postulated hypocalcemia induces anterior ischemic optic neuropathy by causing a decrease in axonal transport, thus leading to axonal and optic disc swelling. This localized effect of hypocalcemia on optic nerve function could explain those cases where the CSF (cerebrospinal fluid) pressure is normal. In the presented case CSF opening pressure was normal, but one month after the onset of the symptoms optic neuritis already occurred. Transitory phase of papilledema was missed. But, the question of the unilaterality remains to be answered.

It is believed that, in case of optic neuritis, the correction of serum calcium is insufficient for the recovery of visual function. As in myocarditis event [5,10,11], a restoration of the intracellular calcium is more important for the recovery of the optic nerve than serum calcium concentration which takes some amount of time. The patient's visual acuity improved from 6/192 (1.5 logMAR) to 6/30 (0.7 logMAR) after 2 years. It is believed the visual acuity loss in the presented case was due to low intracellular calcium during the cataract surgery. The mechanism of origin of the optic neuritis in hypocalcemia has not been clarified yet.

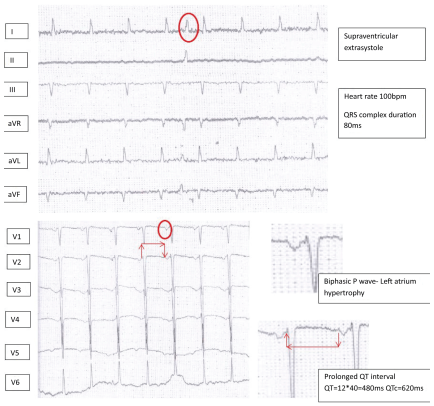

Figure 1: Hypertrophy of the left atrium and ventricle, negative T-wave in V4 – V6, prolonged QT interval- 480 ms.

	11/2009	12/2009	01/2010	04/2010	11/2010	01/2011	05/2011	08/2011	01/2012	06/2012	02/2013	04/2013	05/2013	Normal
Calcium mmol/L	1.03	1.37	2.21	2.12	1.99	1.9	2.37	2.31	2.02	1.82	1.48	1.22	1.01	2.15-2.65
Ionised Ca mmol/L	0.49	0.62	1.08	1.19	1.20	1.1	1.18	1.14	1.13	0.9	0.67	0.75	1.02	1.12-1.32
Vitamin D ng/ml	3.9	4.1	5.5	6.3	4.3	5.5	4.45	6.5	6.6	7.9	8.6	8.5	10.5	<10 low; 10-30 insuff. >30 good
Inorganic phosphate mmol/L	2.72	2.6	2.47	1.8	1.73	2.05	1.52	1.71	1.74	2.03	2.39	2.26	1.9	0.8-1.55
Parathormone	<4.0	<4.0	<4.0	<4.0	<4.0	<4.0	<4.0	5.55	5.67	5.23	<4.0	<4.0	5.13	6-10
Visual acuity OD	6/60 1.0logMAR Cataract	6/120 1.3logMAR	6/152 1.4logMAR	6/192 1.5logMAR	6/192 1.5logMAR	L+P+ cataract	6/6 0logMAR Postop.	6/6 0logMAR	6/7.5 0.1logMAR	6/10 0.2logMAR	6/15 0.4logMAR	6/15 0.4logMAR	6/6 0logMAR	
Visual acuity OS	6/76 1.1logMAR cataract	6/152 1.4logMAR	6/192 1.5logMAR	6/6 0logMAR Postop.	6/192 1.5logMAR left vitrectomy	6/152 1.4logMAR	6/152 1.4logMAR	6/60 1.0logMAR	6/76 1.1logMAR	6/76 1.1logMAR	6/76 1.1logMAR	6/120 1.3logMAR	6/30 0.7logMAR	
	11/2009. (No Ca sup.)	12/2009. (with Ca sup.)	8/2010. (with Calcium sup.)				5/2011. (with Calcium sup.)		6/2012. (with Calcium sup.)		5/2013. (with Calcium sup.)			Normal values
Left atrium cm	4.7	4.6	4.1				3.5		3.4		3.3			2.0-4.0cm
Left ventricle EDD* cm	6.6	6.6	6.3				6.1		5.1		5.0			3.9-5.6cm
Left ventricle ESD* cm	5.1	5.1	5.1				4.7		3.4		3.3			<4.0cm
Left ventricle EF* %	37	40	45				53		56		60			64±10%
Mitral valve	1-2/1.4	insuff. 1+	1.1/1.3	insuff.2-3+		0.8/1.1	insuff.2+		0.5/0.9	insuff.1+	0.5/0.9		insuff. 1+	
Tricuspid valve	0.5	insuff. 1+	0.6	insuff. 1+		0.6	insuff. 1+		0.6	insuff. 1+	0.6		insuff. 1+	

Table 2: *EDD -end diastolic dimension; ESD -end systolic dimension; EF -ejection fraction; insuff- Insufficiency; Other measurements which are standardly part of the heart ECHO were within the normal limits and for that reason have not been included in presented table.

This case report showed that administration of the adequate supplement therapy will lead to withdrawal of all neurological symptoms, thus improving the life quality of the patient. It can prevent severe loss of visual acuity due to hypocalcemia induced optic neuritis, or to stabilize and, to a lesser extent, improve visual acuity once it has been damaged. Unilaterality of the process present in the left eye, as well as the exact mechanism of PNO damage, remain unclear. The presented results indicate visual acuity is highly dependent of the serum calcium levels in direct manner.

As for the heart insufficiency due to heart enlargement it was shown that adequate supplement therapy will improve ejection fraction of the heart, as well as cause reduction in the size of the enlarged left heart.

When confronted with the patient without any known endocrinology disease, reporting a wide range of neurological, cardiologic and psychiatric problems accompanied with slightly diminished visual acuity, physicians should be aware of the possibility of hypocalcemia because adequate treatment can prevent severe eye, heart and neurological complications.

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